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N65-88651

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Paper for Journal of Aerospace Medicine

CARDIOVASCULAR RESPONSES TO TRANSVERSELY APPLIED ACCELERATIONS

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ABSTRACT

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A study of the effect of x-axis accelerations on the human body in relation to the arterial blood oxygen saturation as measured with the ear oximetry technique is reported. Preliminary observations as to the effect of EBO ($-G_x$), EBI ($+G_x$), and EBL ($+G_y$) accelerations on venous blood pressure inside the thoracic cavity near the right atrium, and arterial blood pressure peripherally are also presented.

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INTRODUCTION

Responses in the cardiovascular system to sustained acceleration stress applied to the human body at a right angle to the spinal axis and along the x-axis of the body are not remarkable when one considers only the arterial blood pressure and heart rate changes. When one considers the right atrial venous blood pressure, arterial blood oxygen saturation, and cardiac rhythm, then there are some remarkable changes which differ according to how the acceleration force is applied, i.e., EBO ($-G_X$) or EBI ($+G_X$) (ref. 1). The arterial blood oxygen saturation, however, is as much a measure of the pulmonary system efficiency under these stresses as it is an indication of the status of the cardiovascular system.

Reference 2 reported evidence obtained by chest x-rays that there were marked shifts in the distribution of blood in the pulmonary circulation during EBI ($+G_X$) accelerations. Reference 3 similarly reports shunt effects in the pulmonary circulation during EBI ($+G_X$) acceleration and ascribes the cause to circulatory congestion with alveolar collapse or atelectasis. References 4 and 5 pointed out that the alveolar ventilation is reduced during EBI ($+G_X$) acceleration and essentially normal during EBO ($-G_X$) acceleration.

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In the light of the evidence of pulmonary circulation changes and the diminished alveolar ventilation, it is not surprising that there is an arterial oxygen desaturation during EBI ($+G_X$) acceleration.[3,5,6,7,8]

Since the alveolar ventilation has been shown to be normal during EBO ($-G_X$) acceleration,[4] it was reasonable to assume that the arterial oxygen saturation would also remain normal. Some preliminary data substantiating this viewpoint are reported in reference 5.

Lindberg, et al.,[8] found a striking increase in right atrial pressure in subjects during EBI ($+G_X$) acceleration. The increase in atrial pressure became greater with a greater magnitude of acceleration and at 5 g reached a value four times that found at 1 g. Again the right atrial pressures were compared during EBO ($-G_X$) and EBI ($+G_X$) accelerations. It was felt that since the causes for the increase in right atrial pressure during EBI acceleration (that of an increased blood volume in the thorax together with the compression of the thorax and decreased respiratory excursions of the thorax and diaphragm) do not appear during EBO acceleration, venous pressure should increase very little.

It was of additional interest to explore by means of a few preliminary tests the effects of lateral acceleration (y-axis) on the blood oxygen saturation as well as venous pressures.

The effects of transverse acceleration on cardiac rhythm has been previously described in reference 5.

METHODS

The blood oxygen saturation during transverse acceleration is measured with the same degree of accuracy by means of the ear oximeter as it is with the cuvette method.* The technique used for both methods is described in reference 9. In this study the housing of the ear oximeter device was modified so that it could be used under the dynamic conditions of acceleration. Calibrations were carried out prior to each run on the centrifuge and the results were recorded on an 8 channel Brush recorder in terms of percent saturation.

To measure the venous pressures a No. 50 polyethylene catheter was introduced into the basilic vein at the elbow of the right arm and passed toward the heart until its tip was in the superior vena cava just outside the right atrium. A Statham pressure transducer was attached to this catheter and located at the level of the superior vena cava so that no hydrostatic head existed. The transducer was calibrated with the recorder to indicate venous pressures in millimeters of Mercury. It was not possible to introduce the catheter in the right basilic vein of the particular subject chosen for the lateral acceleration runs, so the left basilic vein was entered instead, and the tip of the catheter rested in the left innominate vein.

Arterial blood pressures were recorded by means of an autosphygmomanometer device.[10] A photocell device attached to the ear was used to measure relative changes in arterial systolic blood pressure in lieu of the ear oximeter device during the lateral acceleration runs.

The tests were conducted on the University of Southern California human centrifuge equipped with a two-position support, the pilot restraint system of reference 11 and the physiological instrumentation system of reference 12 (fig. 1).

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*Based on a private communication from Dr. E. H. Wood, Dept. of Physiology, Mayo Foundation.

The length of the data runs (from 1.0 to 1.5 minutes) was dependent on an assessment of the data as it appeared on the recorder at the operator's station.

Vectorcardiograph studies were made using a special vectorcardiograph computer and a Sanborn recorder. This computer was constructed by the Stanford Research Institute under NASA Contract NAS2-526. The data obtained will be the subject of a separate publication.

A total of 8 subjects was used in this study: 6 were qualified test pilots; the other two, used primarily for the venous blood pressure studies, were medical students.

RESULTS AND DISCUSSION

Oximetry

The results of the oximetry studies under EBI ($+G_X$) accelerations verified the findings of previous investigations. At the 4 g level EBI ($+G_X$) only small increments of desaturation were demonstrated. However, at 6 g EBI ($+G_X$) the oxygen saturation decreased from around 96 percent at the onset of acceleration to as low as 71 percent after 70 seconds at maximum acceleration. Table I illustrates the ranges of desaturation in the 8 subjects for both EBI ($+G_X$) and EBO ($-G_X$) acceleration. During EBO ($-G_X$) acceleration the arterial blood oxygen saturation increased in two subjects, probably because of a tendency to hyperventilate mildly during EBO ($-G_X$) acceleration.

It should be mentioned that quite frequently the chest support of the anterior restraint (ref. 11) is fastened too tightly over the subject. This causes a restriction of the thoracic cage and a diminished vital capacity and tidal volume even at 1 g. As a result during EBO ($-G_X$) acceleration a mild arterial blood oxygen desaturation could and did take place. This can be avoided through more careful fitting and adjustment of the anterior support of the restraint system.

Figure 2 is a time history of subject J. E. showing the arterial blood oxygen saturation during runs of 6 g EBO ($-G_X$) and 6 g EBI ($+G_X$).

Venous Blood Pressure

Because of the limited number of subjects available for the venous pressure studies under EBO ($-G_X$) and EBI ($+G_X$) (2 subjects) only preliminary data can be submitted, and because of difficulty in entering the right basilic vein of one subject, only data from one subject is submitted. The subject D. W. had the catheter inserted in the left basilic vein with its tip located in the left innominate vein. The location of the catheter tip in subject S. R. was such that no variation in the 1 g venous pressure was found to occur when his position was changed from 1 g normal to 1 g EBO ($-G_X$) and 1 g EBI ($+G_X$).

Figure 3 is a time history of the venous blood pressure measured with the catheter tip in the superior vena cava just outside the right atrium of the heart in subject S. R. At 4 and 6 g EBO ($-G_X$) there is only a slight (5 mm of Hg) increase in venous pressure as compared to 1 g. During 4 and 6 g EBI ($+G_X$) increases of approximately 20 mm are shown over the 1 g level. The marked elevation in the SVC pressure just before the onset of acceleration for the 6 g EBO ($-G_X$) run was caused by an inadvertent Valsalva maneuver by the subject.

It is admittedly hazardous to venture an opinion from the results of a single series of EBO and EBI runs using just one subject. Further investigations are needed to determine the effect of these two differently applied transverse accelerations on various blood pressures at or near the right atrium, but it is interesting, however, to note the trend in this one subject.

Lateral Acceleration Studies

As previously stated the photocell ear pulse device was substituted for the ear oximeter during the lateral acceleration studies on subject D. W. This device demonstrated the usual rise in arterial pressure during transversely applied accelerations along the x-axis of the body. These changes as measured by this device are expressed as relative increases in blood pressure since it is not possible at this time to calibrate the device for absolute values. Figure 4 is a time history of the acceleration and the changes in relative arterial systolic blood pressure measured in mm of Hg during a 4 g EBL ($+G_y$) run. Figure 5 is a time history of the venous blood pressure in the same subject measured with the catheter tip in the left innominate vein during 2, 3, and 4 g EBL runs.

Remarkable changes in both systolic arterial pressure and venous pressure are demonstrated in the subject during the 4 g EBL ($+G_y$) acceleration. These changes demonstrate the extreme sensitivity of the cardiovascular system to lateral accelerations and the possible detrimental effects.

There has always been abundant evidence that the cardiovascular system with its fluid medium is extremely vulnerable to the increased forces of gravity during sustained moderately high acceleration stress. The effective remedy for this vulnerability was considered to be the proper orientation of the body in the acceleration field, and the rise in arterial blood pressure, which is seen when the body is oriented with its x-axis in the direction of the inertial forces, would suggest that this orientation is advisable. Little consideration, however, has been given to the possibility of detrimental effects of this orientation on both cardiac rhythm and venous pressures if the direction of the application of these forces is not considered adequately. That EBI ($+G_X$) acceleration causes increases in intrathoracic and right atrial pressures has been amply shown. That EBO or ($-G_X$) acceleration does not cause this same increase in venous pressure is suggested by the one subject reported in this study. The fact that premature ventricular contractions are frequent during exposure to EBI ($+G_X$) and rare during EBO ($-G_X$) acceleration would also indicate the necessity for qualifying the direction of the x-axis tolerance to acceleration more specifically (ref. 1).

Lateral accelerations, i.e., those applied transversely to the spinal axis of the body along the y-axis, apparently cause marked alterations which are detrimental to both arterial and venous pressures. This type of applied acceleration should receive more thorough investigation in the future.

SUMMARY AND CONCLUSIONS

Cardiovascular function during acceleration applied transversely to the spinal axis of the body varies according to the direction of the application of the inertial forces. It is quite well established that the arterial blood oxygen saturation is diminished markedly during EBI ($+G_X$) accelerations. Likewise, it is apparent that the venous blood pressure in the thorax at the level of the superior vena cava and the right atrium is elevated about 20 mm of Hg over that found at 1 g when the individual is subjected to 4 to 6 g EBI ($+G_X$) acceleration. When the acceleration stress is of the EBO ($-G_X$) variety, the arterial blood oxygen saturation is not abnormal and the venous pressures on the superior vena cava and right atrium are not elevated.

Lateral accelerations of the EBL ($+G_y$) variety probably cause a decrease in the arterial as well as the venous blood pressure.

Cardiac rhythm irregularities in the form of premature contractions, often originating in the ventricle, are more frequently the result of EBI ($+G_x$) acceleration and are rarely seen during EBO ($-G_x$) accelerations.

REFERENCES

1. Smedal, Harald A., Creer, Brent Y., and Wingrove, Rodney C.: Physiological Effects of Acceleration Observed During a Centrifuge Study of Pilot Performance. NASA TN D-351, 1960.

2. Hershgold, E. J.: Roentgenographic Study of Human Subjects During Transverse Accelerations. Aerospace Medicine, 31:213-219, 1960.

3. Barr, P. O.: Hypoxemia in Man Induced by Prolonged Acceleration, Acta Physiologica Scandinavica 54:128-137, 1962.

4. Rogers, Terence A., and Smedal, Harald A.: The Ventilatory Advantage of Backward Transverse Acceleration. Aerospace Medicine, 32:737-40,1961.

5. Smedal, Harald A., Rogers, Terence A., Duane, Thomas D., Holden, George R., and Smith, Joseph R., Jr.: The Physiological Limitations of Performance During Acceleration. Aerospace Medicine, 34:48-55, 1963.

6. Steiner, Sheldon H., and Mueller, Gustave C. E.: Pulmonary Arterial Shunting in Man During Forward Acceleration. J. Appl. Phys., 16:1081-6, 1961.

7. Mueller, Gustave C. E.: Cardiovascular Effects of Forward Acceleration in Bio-Assay Techniques for Human Centrifuges and Physiological Effects of Acceleration, Ch. XI, P. Bergeret, ed., Pergamon Press, New York, 119-129, 1961.

8. Lindberg, Evan F., Marshall, Hiram W., Sutterer, William F., McGuire, Terence F., and Wood, Earl H.: Studies of Cardiac Output and Circulatory Pressures in Human Beings During Forward Acceleration. Aerospace Medicine, 33:81-91, 1962.

9. Wood, Earl H., Sutterer, William F., and Cronin, Lucille: Oximetry. Medical Physics, 3:416-445, 1960.

10. Smedal, Harald A., Holden, George R., and Smith, Joseph R., Jr.: A Flight Evaluation of an Airborne Physiological Instrumentation System, Including Preliminary Results Under Conditions of Varying Accelerations. NASA TN D-351, 1960.

11. Vykukal, Hubert C., Gallant, Richard P., and Stinnett, Glen W.: An Interchangeable, Mobile Pilot-Restraint System, Designed for Use in High Sustained Acceleration Force Fields. Aerospace Medicine, Vol. 33, March 1962, pp. 279-285.

TABLE I.- ARTERIAL BLOOD OXYGEN SATURATION DATA ON 8 SUBJECTS AT 1 g AND 6 g EBO ($-G_x$) AND EBI ($+G_x$)

Subject	Blood O_2 percent saturation at 1 g	Blood O_2 percent saturation at 6 g	
		EBO	EBI
J.E.	94.5	93	74
G.H.	96.0	86	77
G.N.	96.0	88	71
S.R.	95.5	100	81.5
R.I.	96.0	100	91.5
D.W.	98.0	99.5	86.5
R.G.	99.0	87.0	81.5
G.S.	96.0	97.5	79.0

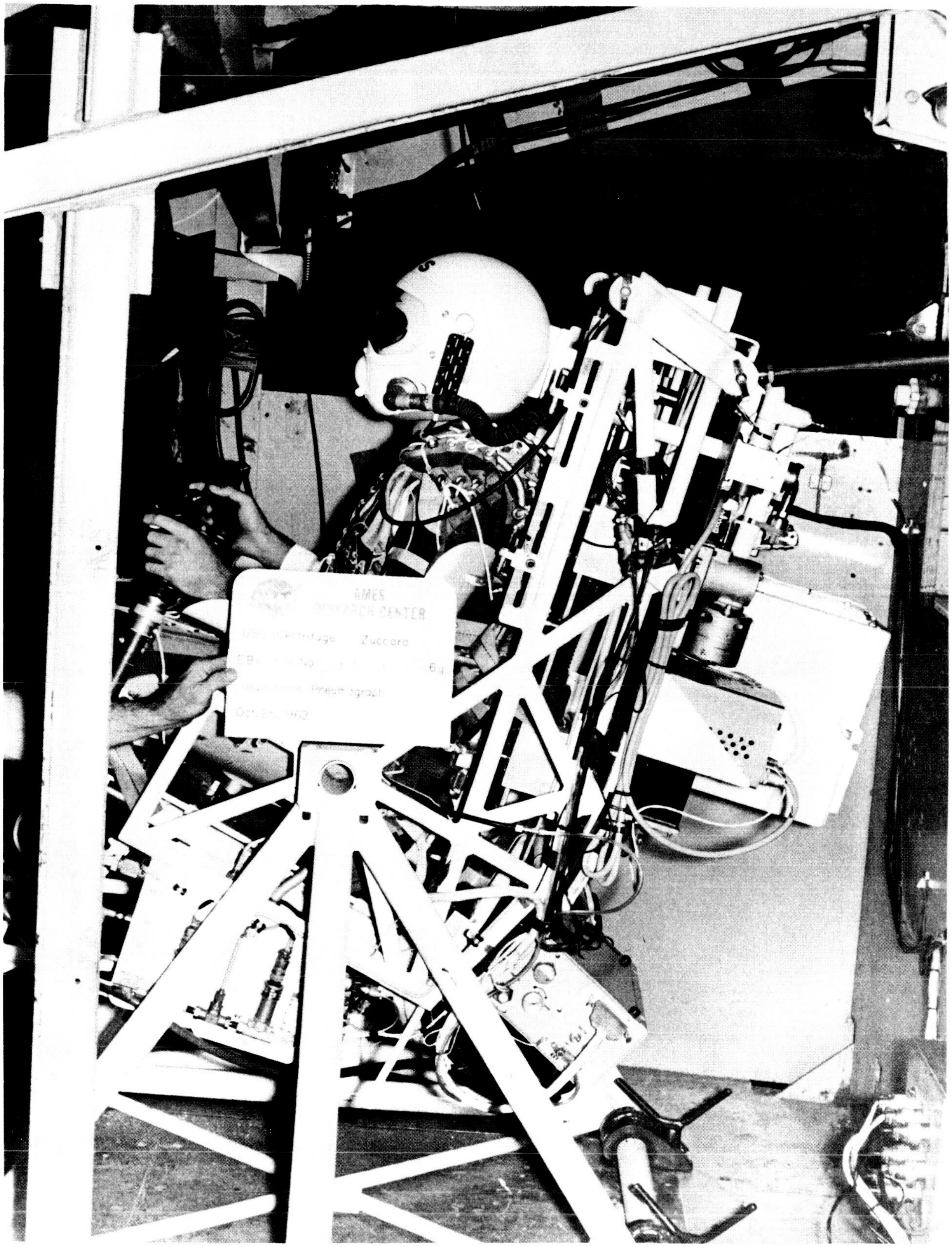

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Figure 1.- Subject in restraint system ~~prior to run at U.S.R.~~

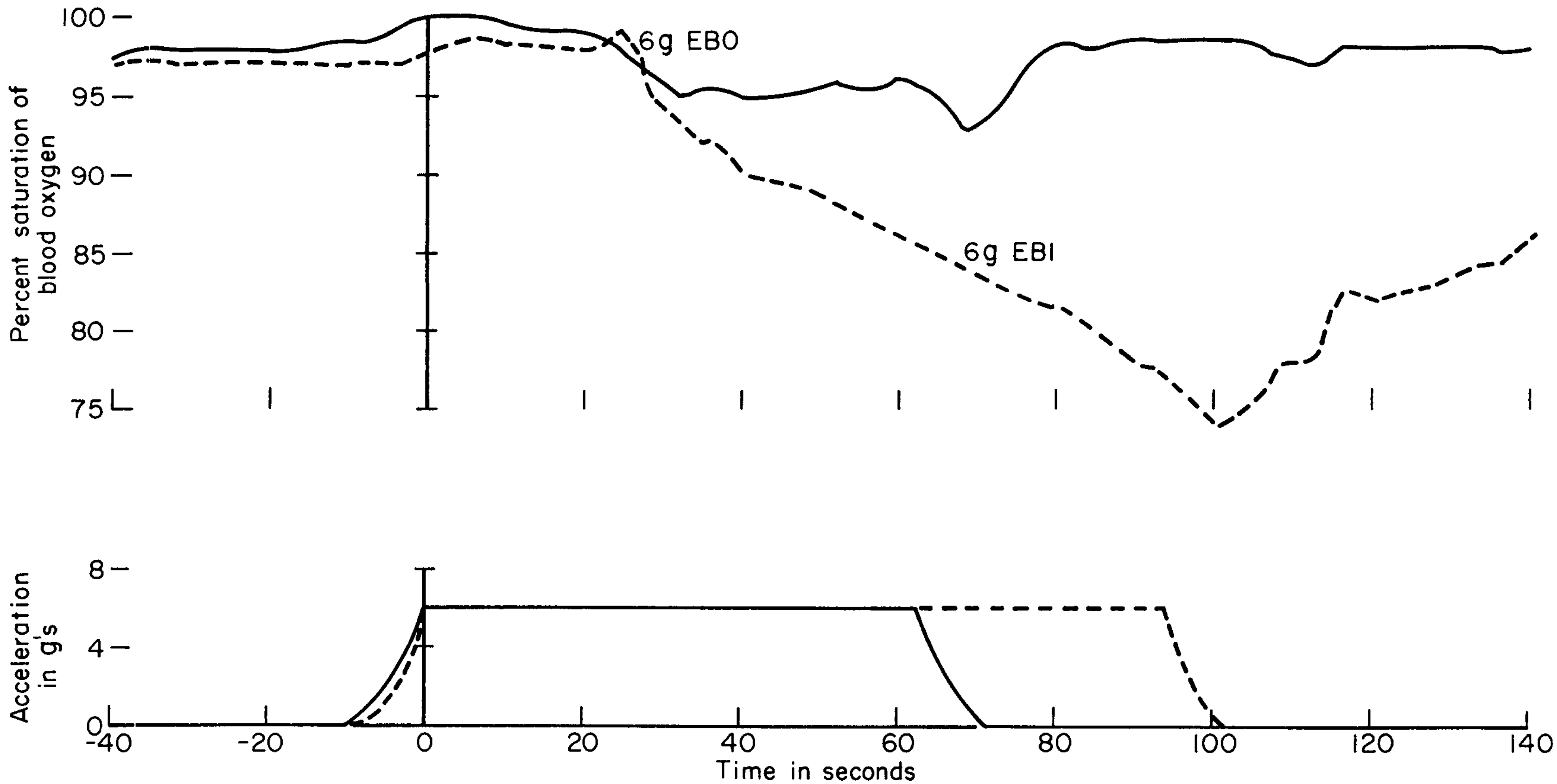

Figure 2.- Arterial blood O_2 saturation in subject J. E.

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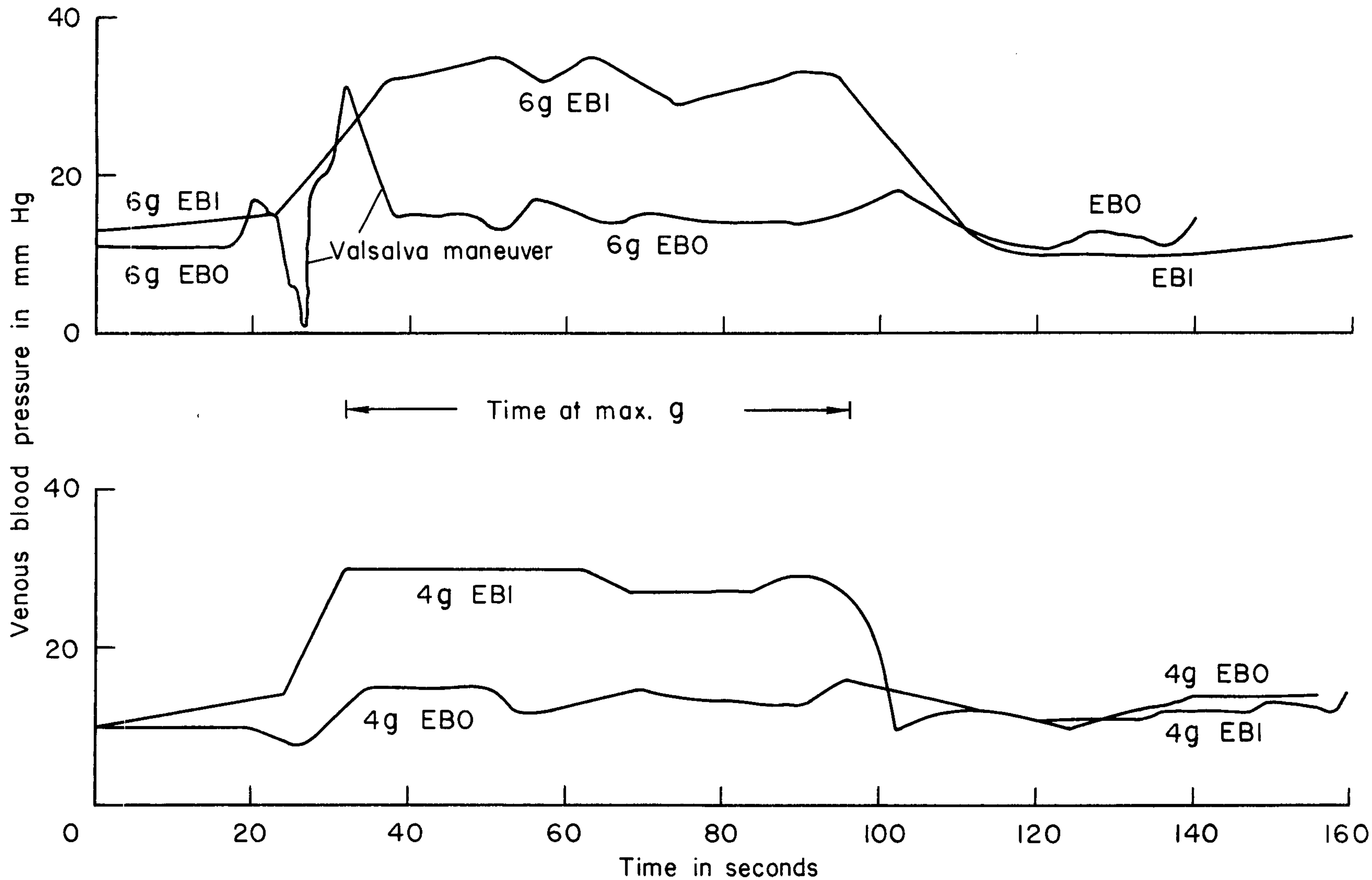

Figure 3.- Time history of subject S. R. showing superior vena cava blood pressure changes at 4 and 6 g EBI and EBO.

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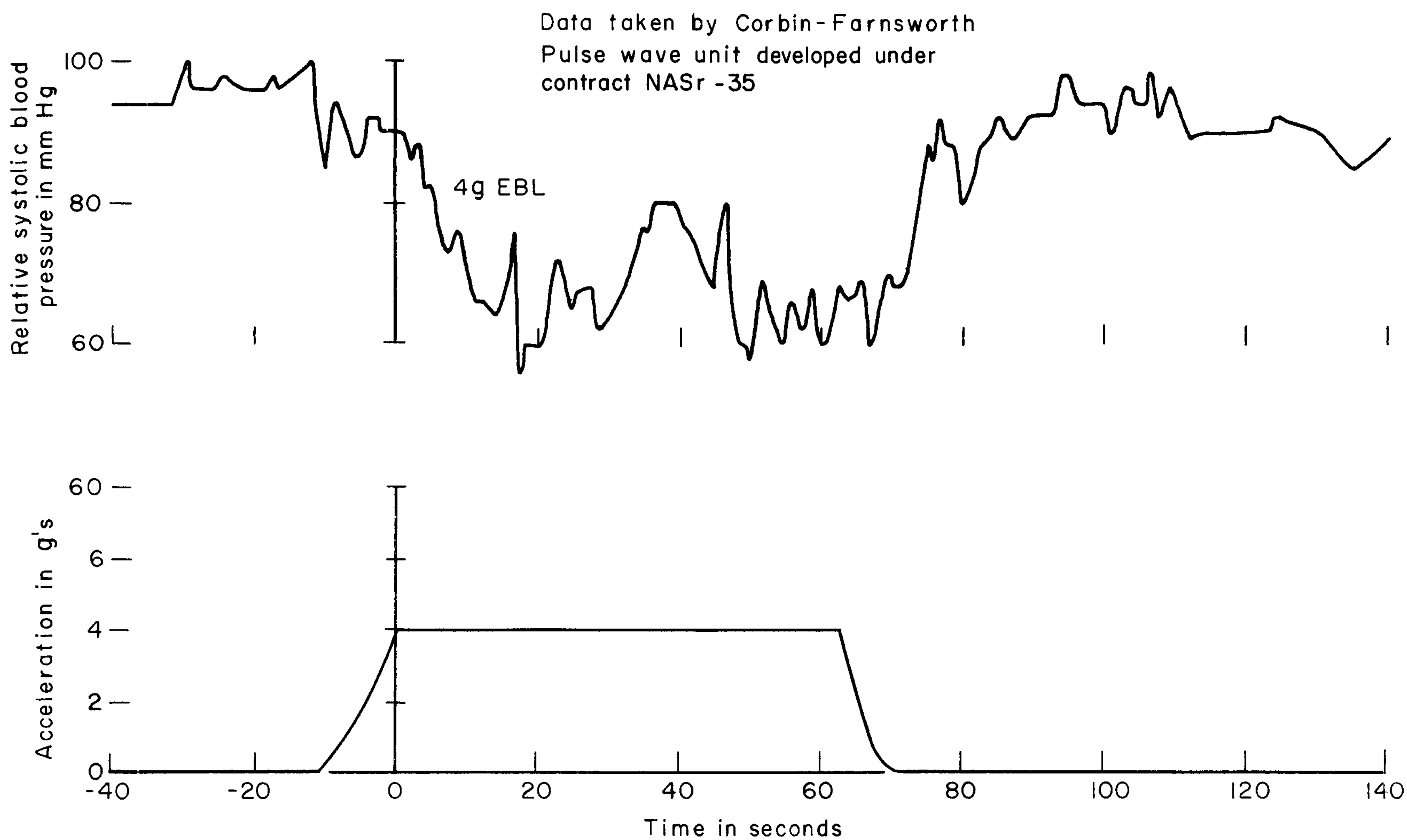

Figure 4.- Relative arterial systolic blood pressure at the ear level during EBL ($+G_y$) acceleration.

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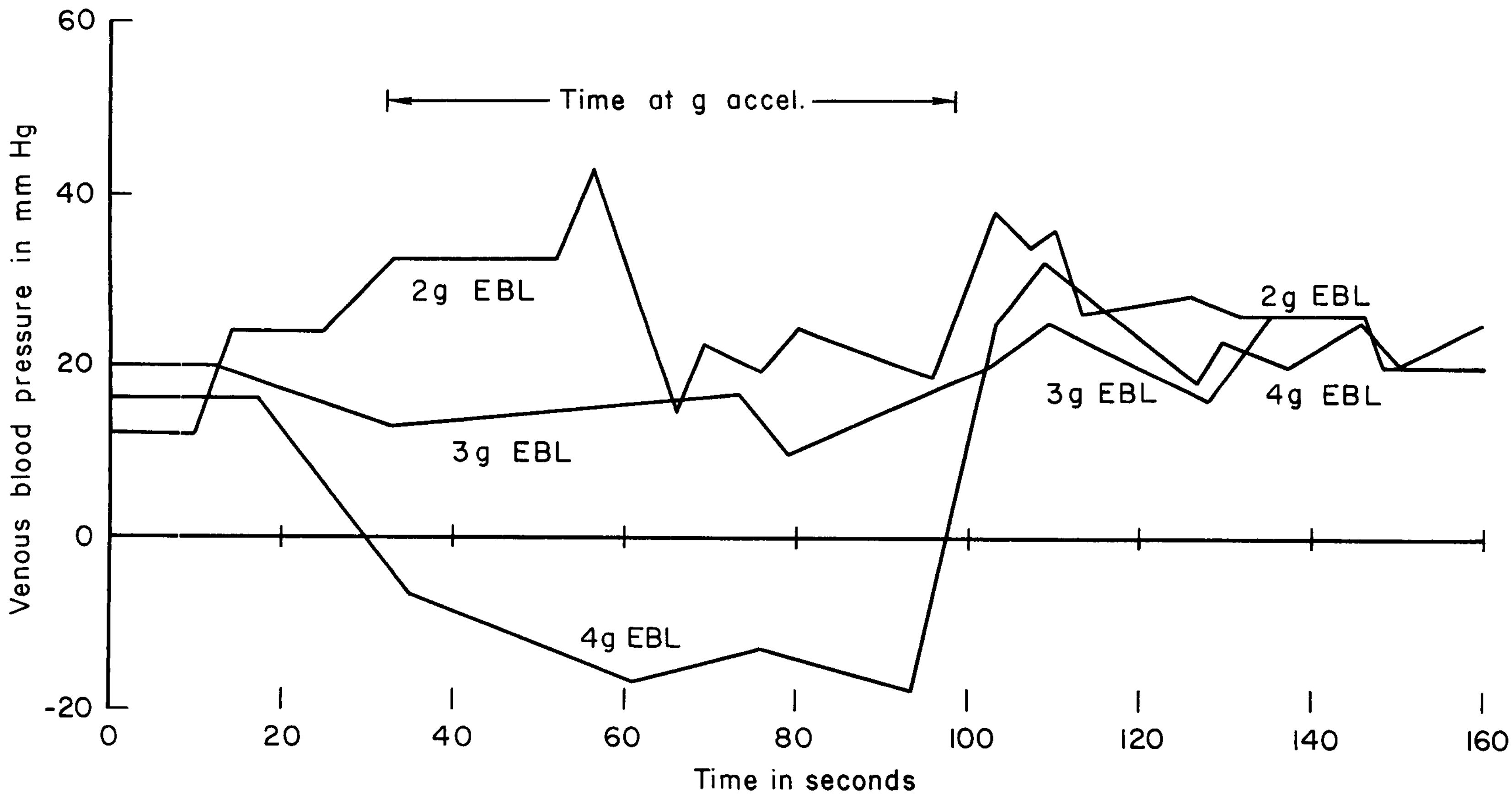

Figure 5.- Time history of venous blood press in the left innominate vein during EBL ($+G_y$) acceleration.

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